T Trilinolein preserves the ultrastructure of

mitochondria in isolated rat heart subjected to global

ischemia through antioxidant activity as measurement

by chemiluminescence.

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摘要

Abstract

Oxygen-derived free radicals (OFR) have been proposed as the cause of myocardial damage through lipid peroxidation during ischemia and reperfusion. Antioxidants can effectively ameliorate the damage induced by lipid peroxidation. Trilinolein is a triacylglycerol recently purified from the well known traditional Chinese herb Panax pseudoginseng, which has been used in treating circulatory disorders among Chinese for hundreds of years; it has linoleate as the only fatty acid residue in all three esterified positions of glycerol. This chemical has recently been demonstrated to have antioxidant activity by enhanced chemiluminescence. The addition of phorbol myristic acetate (PMA) to medium containing leukocytes produces OFR; this phenomenon was measured by chemiluminescence. Addition of trilinolein to medium containing leukocytes preceding the addition of PMA suppressed the production of OFR. The control value of chemiluminescence of a medium containing leukocytes with addition of PMA was 9.23 +/- 1.19 x 10(3) mV. The most effective concentration of trilinolein was 10(-7) mol/l which decreased the signals to $4.59 \pm 0.02 \times 10(3) \text{ mV}$ (p < 0.001). The antioxidant effect had a concentration-response curve similar to alpha-tocopherol. After pretreatment for 15 min with trilinolein at a concentration of 10(-7) mol/l in isolated perfused rat heart which had been subjected to 60 min of global ischemia, the integrity of the rat heart mitochondria was preserved as examined under the electron microscope. No swelling of mitochondria occurred and there was good alignment of cristae and absence of amorphous density. Previous experiments have shown that trilinolein can also improve erythrocyte deformability in vitro. Infarct size reduction of about 50% was also demonstrated in in vivo rat heart subjected to 4 h coronary occlusion. The mechanism of myocardial protection, in addition to the antioxidant effect, is suggested as maintaining the membrane fluidity of cardiomyocytes

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